PRESSURE BREATHING WITH OXYGEN AND HELIUM OXYGEN IN PULMONARY EDEMA AND OBSTRUCTIVE DYSPNEA*

HYLAN ARTHUR BICKERMAN and GUSTAV JULIUS BECK

THE treatment of pulmonary edema has always presented a serious challenge to the attending physician. The increasing incidence of this disease is due in part to an aging population in which heart disease is prevalent and to the increasing use of toxic gases in industry and war. However, this clinical entity is frequent during the course of other serious illnesses, such as atypical and virus pneumonia, shock, and bulbar poliomyelitis.

The use of inhalation of gases under positive pressure for the treatment of obstructive dyspnea and acute pulmonary edema was developed independently by Poulton[1] in England and Barach[2] in this country in 1936. Historically, it is of interest that Oertel[3] in 1878 used compressed air in the treatment of a patient with severe asthma. In 1896, Norton[4] reported a case of pulmonary edema due to carbolic acid poisoning which responded rapidly to the application of forced respiration under positive pressure. Several years later, Haven Emerson[5] and, subsequently, others showed that edema of the lungs produced in rabbits by intravenous adrenalin could be relieved by artificial respiration. Barringer[6] made use of this suggestion in the successful treatment of a patient with cardiac insufficiency who developed acute pulmonary edema.

While inspiratory positive pressure respiration was commonly accepted as a method of resuscitation in accidental asphyxia, continuous pressure breathing was first used clinically, in the treatment of edema and obstructive dyspnea.[7] Later, high altitude flying stimulated intensive studies on the physiologic effects of various forms of pressure breathing.[8] Newer techniques, including cardiac catheterization, have given additional and more exact information on the circulatory and respiratory effects in man.[9]

Pulmonary edema presents a characteristic clinical picture manifested

* From the Research Service, First (Columbia) Division of the Goldwater Memorial Hospital and the Department of Medicine, College of Physicians and Surgeons, Columbia University.

by bubbling rales in the lungs and severe anoxia. The physiologic factors involved in the transudation of serous fluid from pulmonary capillaries into the alveoli depend upon the resultant of forces represented by the hydrostatic pressure within the capillaries, the colloid osmotic pressure, capillary permeability and lymphatic drainage.

Welch[10] in 1878 postulated that a relative insufficiency of the left ventricle resulted in an accumulation of blood in the lesser circulation resulting in severe congestion. In addition to the increased hydrostatic pressure, local areas of anoxia would effect a change in capillary permeability. An increase in capillary permeability may be induced by specific irritants, local inflammation, toxemia and severe anoxia. This factor is important in the pathogenesis of pulmonary edema occurring in the course of pneumonia, peripheral circulatory failure, and gas poisoning.

Other factors which may play a role in edema of the lungs are:

Severe depression of the colloidal osmotic pressure of the blood in certain disease states such as nephrosis and hepatic insufficiency.

Neurogenic and reflex mechanisms occurring as a result of cerebrovascular accidents, trauma to the skull and disease of the central nervous system.[11]

In obstructive dyspnea due to severe asthma or to lesions of the tracheobronchial tree, the lung capillaries are exposed to a high negative intrapulmonary pressure on inspiration which exerts a suction-like action on the capillary walls and tends to further pulmonary congestion.

As a result of the investigations of Barach and his collaborators,[7] it was observed that the use of positive pressure breathing decreased the effort necessary for inspiration with a corresponding decrease in the pathologically elevated negative pressure. In addition, continuous pressure breathing had the following physiologic advantages in the treatment of pulmonary edema and obstructive dyspnea:

Exerted a direct opposing pressure on the external surface of the pulmonary capillaries and counteracted in some measure the elevated hydrostatic pressure.

Served to retard the entrance of blood into the right side of the heart, thereby diminishing the accumulation of blood in the lungs.

Provided greater patency of the tracheobronchial airway, especially during the expiratory cycle.[12]

Pressure Breathing Equipment

There are two general types of pressure breathing, 1) intermittent and 2) continuous. In the former, the major part of pressure is applied during expiration (expiratory pressure breathing) or during inspiration (inspiratory pressure breathing).

1.(a) *Expiratory positive pressure* may be applied with relatively simple devices requiring the patient to exhale against a resistance and thereby increase the intrapulmonary pressure during expiration. Inspiration takes place at the customary negative pressure. The Meter mask[13] is equipped with a metal disc in front of the expiratory flutter valve, in which expiration takes place through a restricted orifice, the size of which regulates the pressure. Greater accuracy in the control of pressure is obtained when expiration is conducted through a water bottle calibrated in centimeters.[14,15]

The use of positive pressure during expiration is useful in the treatment of acute pulmonary edema as a result of alterations in permeability of the pulmonary capillaries, and left ventricular failure. It is customary to begin treatment with an expiratory pressure of 5 cm. of H_2O, which is gradually lowered to atmospheric pressure when improvement is manifested. An oxygen concentration of from 40 to 60 per cent is employed. Expiratory positive pressure is also used to prevent or control the oozing that takes place from the tracheobronchial tree following tracheotomy. When this is used, the tracheotomy tube is prolonged so that expiration proceeds through a valve into a water bottle set at a pressure of 4 to 5 cm. of H_2O, and this is gradually lowered during a period of twelve hours to 1 cm. of H_2O.[16]

Expiratory pressure breathing results in rather labored expiration and, since there is no aid to inspiration, the breathing effort is greater than with inspiratory or continuous pressure.

(b) *Inspiratory Positive Pressure Breathing.* Gas enters the lungs under positive pressure in inspiration; pressure is then automatically released and expiration occurs as a passive act. Such apparatus is useful in the treatment of patients with depressed or failing respiration as in coma or poisoning. The respirators give adequate pulmonary ventilation when spontaneous respirations have ceased. The essential features of these appliances are a high pressure oxygen tank, a demand valve and a second or "cycling" valve.[17] It should be emphasized that these devices

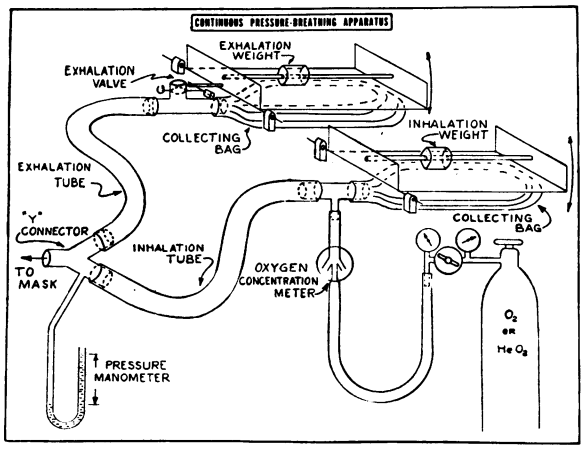

Fig. 1—A double bellows type apparatus which provides continuous positive pressure with either oxygen or helium-oxygen mixtures over the range of 0 to 14 cm. water pressure.

will cycle also with the patient's own respiration following recovery of spontaneous breathing as opposed to the Drinker type respirator.

Since inspiratory positive pressure breathing, conventionally called "intermittent," provides a mean positive pressure during the entire respiratory cycle, it is also effective in the treatment of acute pulmonary edema. A serious defect in its use on a dyspneic patient is the inability of the current models (Pneumolator and Emerson) to provide a rapid immediate volume of inflow. Motley[18] has reported that in some instances the inspiratory velocity of the dyspneic patient will exceed the capacity of the apparatus. The patient inhales against a resistance with a sensation of increased dyspnea or suffocation. This would be especially true for a patient with obstructive dyspnea. In addition, a significant degree of hyperventilation may occur on intermittent positive pressure breathing. A research model with a high inflow capacity (100 L/min.) has been devised but it is not commercially available at present.

2. *Continuous Pressure Breathing.* Pressure is employed in both phases of respiration. The degree of comfort in breathing is much greater than with expiratory pressure breathing alone, and that is especially true when the variation between expiratory and inspiratory pressure is kept at a minimum.

An effective apparatus for the administration of oxygen and helium-

oxygen mixtures is the positive pressure hood.[19] This is useful in the treatment of severe asthma and obstructive dyspnea produced by lesions in the tracheobronchial passageway. Since this is a closed circuit apparatus, it is important to provide efficient cooling and absorption of carbon dioxide with soda lime. Pressures are maintained by a motor blower unit and a pressure control water valve.

A relatively simple bellows device for employing continuous positive pressure has been developed* (Fig. 1). Inspiratory pressure is rapidly built up when oxygen at 15 L/min. is passed through a specially constructed injector meter which delivers 65 liters of gas per minute at an oxygen concentration of 38 per cent to a weighted bag of 2 liters capacity. The expired gases pass into a similarly weighted bag which is equipped with a butterfly type valve. The degree of expiratory and inspiratory pressure is easily adjusted by means of sliding weights. An airtight seal over the face is accomplished by a light plastic Bennett mask with an inflated cuff. With lower rates of inflow, the building up of inspiratory positive pressure is less rapid. When helium-oxygen mixtures are employed, the injector meter is not used. The apparatus is equipped with an emergency inspiratory valve which permits ingress of air if the inspiratory collecting bag should for any reason collapse. The patient is thus protected against breathing under negative pressure. In general, it must be said that inspiratory pressure breathing appeared to us to be somewhat more comfortable than either continuous or expiratory pressure breathing.

The physiologic effects on respiration and hemodynamics of continuous pressure breathing employing the bellows apparatus was studied in eighteen normal subjects at mean mask pressures of 6 to 12 cm. of H_2O. To obtain measurements of the relative cardiac output and stroke volume, we employed a modification of the photo-electric method of ballistocardiography as developed by Dock[20] in which motion of the body is transmitted to a rigid metal bar placed across the shins which is in contact with an occulting plate mounted on frictionless jewelled points. The shadow cast by the occulting edge on a photocell results in changes in electric potential which is recorded by a direct writing electrocardiograph. These records are standard and reproducible with a time lag of less than 0.005 seconds (Figs. 2 and 3).

While the ballistocardiograph, as developed by Starr and others, was

* The bellows type continuous positive pressure apparatus can be obtained from the Oxygen Equipment Manufacturing Co., Norwalk, Conn.

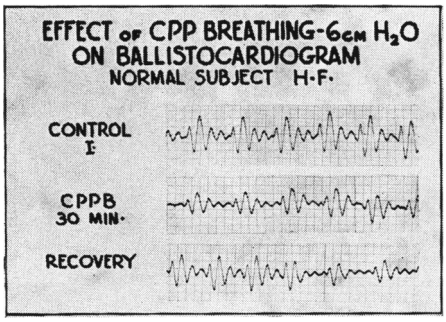

Fig. 2

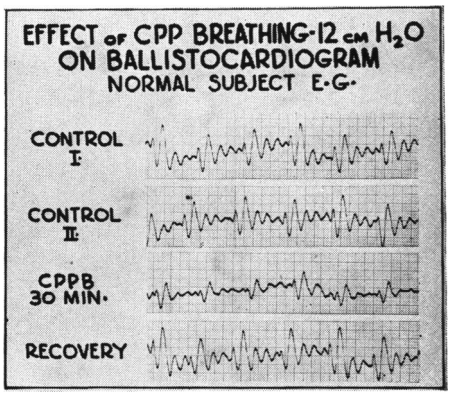

Fig. 3

Figs. 2 and 3—Ballistocardiograph tracings obtained by the Dock method employing a photo-cell pickup.

originally employed to estimate the volume of ventricular ejection, the assumptions made in deriving the formula are numerous, and the reliability of absolute values for stroke volume may be seriously questioned, since the amplitude of the waves depends on ejection velocity rather than volume alone. Since we were interested in relative stroke volumes rather than absolute values, a simplification of Starr's original formula was employed discarding factors K and A.[21]

$$Sv = K\sqrt{(3I + 2J)\,AC^{3/2}}$$

$$\text{Rel } Sv = \sqrt{(I + J)\,C^{3/2}}$$

As Fenn[22] has pointed out, since the ballistocardiograph is sensitive enough to demonstrate the normal respiratory variation in cardiac output, it was felt that any significant changes caused by pressure breathing would be apparent on the tracing and that relative values obtained on the same subject under controlled experimental conditions should be fairly reliable.

In general, there was a slight but variable increase in respiratory rate, pulmonary ventilation and tidal air. In ten subjects at a positive pressure of 6 cm. of H_2O the average increase in pulmonary ventilation was 6 per cent. At 12 centimeters positive pressure, the average increase was 9.5 per cent. These changes were statistically not significant. The relative stroke volumes and cardiac outputs computed from ballistocardiographic tracings showed a significant fall averaging

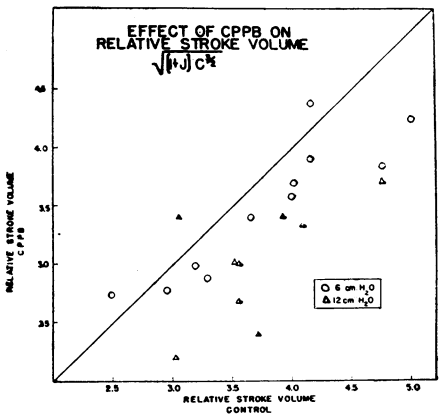

Fig. 4—Graphic illustration of the change in relative stroke volumes; values obtained during a control period at ambient pressure are compared with data on the same subject during a positive pressure period.

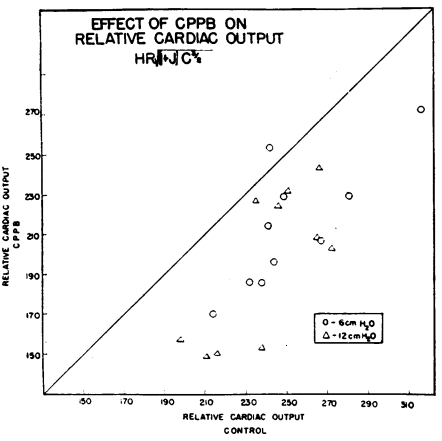

Fig. 5—The relative cardiac outputs while breathing 40% oxygen at atmospheric pressure are compared to continuous positive pressure breathing at 6 and 12 cm. of water pressure.

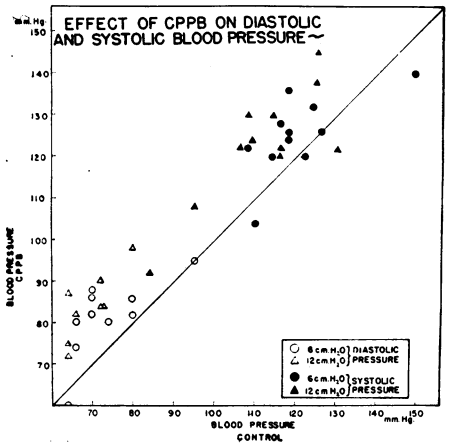

Fig. 6—This comparison graph illustrates the rise in both systolic and diastolic blood pressure accompanying the application of continuous positive pressure.

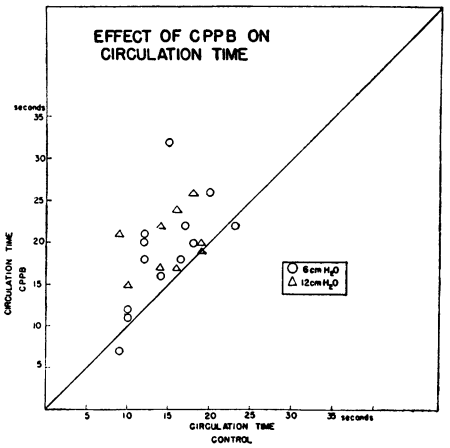

Fig. 7—Circulation time studies with Decholin have been plotted to illustrate the change accompanying continuous positive pressure breathing.

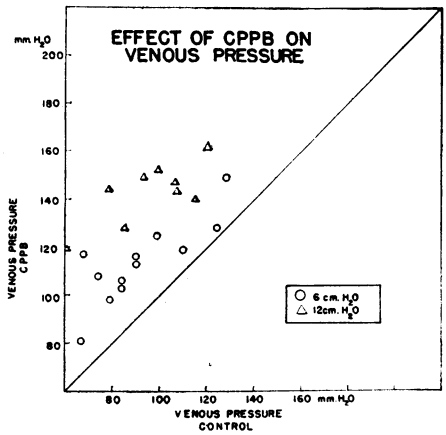

Fig. 8—The striking relationship between the height of the applied pressure and the increase in venous pressure is demonstrated.

6 and 14.6 per cent respectively at 6 cm., and 18 and 20.5 per cent at 12 cm., the magnitude of this decrease being related to the height of the mean mask pressure (Figs. 4 and 5). Minor variations in the heart rate occurred. There was a distinct rise in both systolic and diastolic blood pressure. The elevation in diastolic pressure was greater than in the systolic, resulting in a slight decrease in pulse pressure (Fig. 6). The circulation time for decholin was prolonged (Fig. 7), and the increase in venous pressure was highly significant, with the average elevation being directly proportional to the mean mask pressure (Fig. 8). The hematocrit and plasma protein studies showed no appreciable change from the control level (Table I).

Discussion

These results are on the whole in agreement with previous studies by Barach, Eckman et al,[23] employing continuous pressure breathing in normal, cardiac, and asthmatic patients. Similar results were obtained by Motley and Cournand[24] and Otis and Fenn[22] working with intermittent pressure breathing in the same mean pressure range.

While all observers have reported a consistent fall in the cardiac output of normal subjects with pressure breathing, Cournand remarked on certain differences in the behavior of cardiac patients. In 3 of 9 cardiac patients on a mean mask pressure of 5 to 7 mm. of mercury, the

TABLE I—INFLUENCE OF CPPB UPON HEMODYNAMICS IN NORMAL SUBJECTS

Subject	Mean Mask Pressure cm H_2O	Per Cent Change From Ambient									
		Heart Rate	Stroke Volume	Cardiac Output	Blood Pressure			Venous Pressure	Circulation Time	Hematocrit	Total Proteins
					Systolic	Diastolic	Pulse				
W.M.	6	0.0	— 8.0	— 8.0	— 1.6	+ 8.7	—16.7	+27.8	+113.3	0	+4
D.W.	6	— 3.6	—19.5	—22.5	+11.4	+22.9	— 8.7	+26.2	+29.4	0	+3
G.M.	6	+12.2	— 6.1	+ 4.5	+ 6.8	+ 2.5	+15.8	+ 8.2	—22.2	0	
E.G.	6	— 4.5	— 4.1	—11.2	+ 5.1	+22.9	—20.8	+ 0.8	+75.0	0	—3
M.L.	6	—17.3	— 6.0	—21.8	+ 6.4	+25.7	—18.5	+21.0	— 4.3	0	+7
L.E.	6	—18.4	+ 5.3	—13.9	+ 5.3	+17.1	—13.6	+16.4	+11.1	+4.4	+7
H.F.	6	— 3.6	—15.5	—18.5	+13.0	+12.1	+14.3	+20.9	+30.0	0	—7
M.C.	6	—10.3	—13.0	—19.8	— 5.5	— 4.2	— 4.3	+22.9	+28.0	—2.3	—7
S.B.	6	— 8.1	— 9.4	—19.7	+15.3	+17.5	+ 7.7	+64.3	+14.3	0	+7
I.P.	6	—16.3	+10.0	—20.6	— 2.8	0.0	— 8.2	+45.9	+ 2.9		
J.J.	6	— 3.1	— 5.8	— 8.6	0.0	+ 9.4	—13.1	+22.8	+50.0	—4.7	+5
R.B.	6				+ 3.7	+ 8.1	0.0	+17.9	+67.9	0	
R.C.	6				+ 9.6	+18.2	— 5.3	+47.0		0	
H.S.	6				— 3.3	+15.6	—24.1	+38.9	+20.0		
Average		— 6.6	— 6.0	—14.6	+ 4.5	+12.6	— 6.8	+27.2	+31.9	—0.3	+0.45
I.F.	12	— 2.9	—27.2	—29.4	— 6.2	+ 9.5	—34.8	+33.7	+57.2	0	0
E.G.	12	+ 0.0	—21.8	—21.5	+ 5.2	+16.7	—13.6	+34.2	+133.3	0	—6
M.L.	12	— 8.9	—32.9	—25.4	+13.8	+33.9	—15.9	+50.6	+ 5.3	+4.3	—4
E.R.	12	— 7.1	—15.0	—20.7	+15.1	+15.1	+15.2	+112.2	+50.0	+2.3	+3
S.E.	12	— 8.2	—24.3	—30.6	+13.7	+12.5	+16.2	+55.1	+ 6.2	+5.3	+7
F.H.	12	—12.8	+11.8	— 3.4	+ 8.7	+22.5	—14.9	+39.4	0.0	0	0
J.M.	12	+ 6.2	—13.0	— 7.6	+20.4	+25.0	+11.1	+23.5	+21.4	0	0
W.Mc.	12	0.0	—35.3	—35.7	+14.0	+25.0	— 4.8	+60.2	+44.4	+7.0	+9
W.M.	12	+ 2.9	—11.4	— 9.8	+ 3.5	+24.2	—24.0	+84.6	+50.0	0	—9
R.B.	12				+16.0	+17.2	+14.8	+101.7	+20.0	0	—2
R.C.	12				+10.5	0.0	+22.2	+56.9	+77.9		
H.S.	12				+ 3.3	+ 4.2	+ 2.1	+40.8	+11.1		
Average		— 3.4	—18.0	—20.5	+ 9.8	+17.2	— 2.2	+57.7	+39.7	+1.9	—0.8

cardiac output rose an average of 12 per cent. Lars Werko[25] employing the same pressures, obtained a rise in cardiac output of 10 to 35 per cent in two patients in cardiac failure. This may be related to a decrease in filling pressure of the right auricle, permitting the ventricle to empty more completely, and correlates well with McMichael's study of cardiac output in heart failure in which the application of tourniquets to the extremities diminished the venous return and increased the cardiac output, in accordance with Starling's law of the heart.

In reviewing the literature published since 1935, a total of sixty-four case reports were collected in which positive pressure breathing had been employed for pulmonary edema or obstructive dyspnea. Of these, twenty-eight were in pulmonary edema due to acute left ventricular failure; fourteen suffered from status asthmaticus or obstructive dyspnea; thirteen had pulmonary edema secondary to respiratory depression; six were patients with an extensive pneumonic process complicated by pulmonary edema. There was an interesting case of lung edema due to chlorine gas inhalation[26] and two cases reported by Ansbro[27] of pulmonary edema following tracheotomy. In addition, Carlisle[28] reported 316 patients treated with positive pressure who had inhaled toxic fumes. Most of his patients were seen in the so-called "latent" period prior to the onset of frank pulmonary edema.

Most of the patients observed were the worst possible risks and had received without apparent benefit the commonly used medical measures prior to instituting pressure breathing, which was tried as a last resort. Positive pressure respiration should be administered cautiously in shock in which the venous return to the heart is already retarded. The increased intrapulmonary pressure of from 2 to 4 cm. of H_2O may further diminish the flow of blood into the right auricle. When peripheral circulatory failure is suspected, the blood pressure should be followed at 15 minute intervals and positive pressure discontinued if a fall greater than 10 mm. of mercury takes place in the systolic pressure following the application of pressure breathing.

The usefulness of substituting helium for nitrogen in the gas mixture is due to the lower specific gravity of helium.[29] This property makes it possible to breathe through constricted pulmonary airways at almost one-half the effort required for the respiration of air or pure oxygen, thereby lowering the elevated negative pressure within the chest that occurs in obstructive dyspnea.

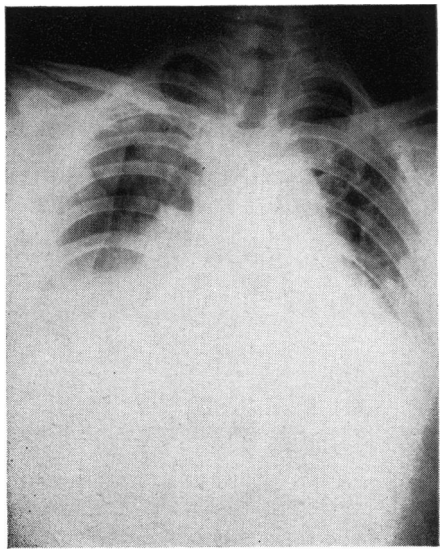

Fig. 9—Portable chest roentgenogram taken on April 26 prior to institution of positive pressure breathing. A major portion of both lung fields is radio-opaque.

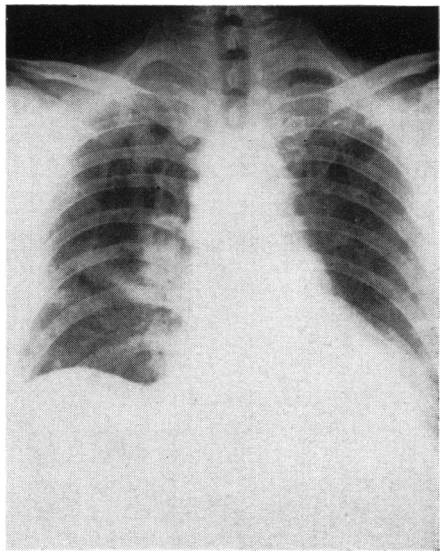

Fig. 10—Portable film taken April 30, after 72 hours of continuous pressure breathing at eight cm. water pressure employing a 65 per cent helium 35 per cent oxygen mixture. Considerable clearing has taken place at both bases.

Case Reports

It is apparent that the primary illness in which pulmonary edema occurs as a complication is frequently of such severity that an ultimate recovery cannot always be expected. However, the clearance of lung edema may allow a seemingly moribund patient opportunity to recover from the original disease. Brief case reports of three subjects are presented:

Case 1: J. K., a 44 year old physician, was admitted to the hospital on April 24, 1947 in a semi-conscious state suffering from smoke poisoning after he had attempted to evacuate patients from a burning building. He regained consciousness in an oxygen tent and appeared relatively comfortable for a period of 24 hours. On the 26th of April, he lapsed into semi-coma, became dypneic and cyanotic and temperature spiked to 103°. He was rapidly digitalized, giving aminophyllin and plasma intravenously, and penicillin therapy was instituted. Auscultation revealed the coarse bubbling rales of pulmonary edema. Bronchoscopic examination was a failure because of the marked edema and friability of the tissues. That night he appeared to be moribund. At 8:00 p.m. continuous positive pressure respiration was applied with a mixture of 65% helium, 35% oxygen at a mean mask pressure of 6 cm. of water. The patient's color immediately improved. Respiratory rate fell from 56/min. to 26/min. and within 45 minutes he

regained consciousness with evidence of pulmonary edema having disappeared. He was maintained on positive pressure for 96 hours, since attempts to decrease the pressure resulted in the reappearance of obstructive dyspnea. His clinical course was stormy, being complicated by a pneumonic process of the right, middle and lower lobes with atelectasis of the left lower lobe. He made a complete recovery and was discharged May 13th (Figs. 9 and 10).

Case 2: J. L., a 58 year old man with a 7 year history of hypertensive cardiovascular disease, complained of severe epigastric and chest pain on July 1, 1949. Clinical and laboratory evidence suggested the diagnosis of a dissecting aneurysm of the abdominal aorta. His course was gradually downhill; further episodes of pain suggested extension of the dissecting process. In addition, he had a severe refractory anemia and marked renal insufficiency. On November 7, severe dyspnea and the physical signs of pulmonary edema were elicited. He received digitalis, morphine and mercurial diuretics with no benefit. That evening he was placed on continuous pressure breathing with oxygen at 6 cm. of water pressure. This cleared the coarse bubbling rales within 15 minutes, but rales would recur when the mask was removed. Positive pressure was maintained until the patient's death on the following day.

Case 3: J. B., a 71 year old woman has had hypertension and diabetes for 15 years. Her diabetes was controlled with small daily injections of protamine zinc insulin, while digitalis and aminophyllin served to keep her compensated. While sitting in a chair, she had a sudden episode of severe dyspnea, and examination 30 minutes later revealed the characteristic picture of pulmonary edema. She was immediately placed on expiratory positive pressure breathing with oxygen at 4 cm. of water pressure, and within 45 minutes all evidence of lung edema had cleared. She made an uneventful recovery, and a follow-up two years later showed no further recurrences of pulmonary edema.

Summary

Physiologic studies were carried out on continuous pressure breathing. When compared to inspiratory pressure, employing a type one or type two curve, no differences in cardiac output or in venous pressure were discerned, provided the mean mask pressure of the intermittent type corresponded to the mean of the continuous pressure breathing.

Pressure breathing, employing either continuous or intermittent pressure, is effective in the treatment of acute pulmonary edema. In the control of obstructive dyspnea due to laryngeal obstruction or to asthma, pressure during inspiration, provided either by continuous or inspiratory pressure breathing, was clinically more valuable than expiratory pressure breathing alone.

An apparatus providing continuous pressure breathing is described, in which a mask is employed instead of a hood, the pressure during inspiration and expiration being produced by suitable weights on the oxygen collecting bags. This apparatus has the advantage of providing an adequate flow of oxygen-enriched atmosphere even in the presence of severe dyspnea, in which the present models employing intermittent

inspiratory pressure have been found to yield an insufficient velocity of air flow.

The case histories of three patients with acute pulmonary edema are reported in which the application of continuous pressure breathing was followed by disappearance of lung edema.

REFERENCES

1. Poulton, E. P.: Left-sided heart failure with pulmonary edema, its treatment with the "pulmonary plus pressure machine," *Lancet,* 1936, *2*:981.
2. Barach, A. L. Use of helium in the treatment of asthma and obstructive lesions in the larynx and trachea, *Ann. Int. Med.,* 1935, *9*:739.
3. Oertel, M. J. Respiratory therapeutics, in Von Ziemssen's *Handbook of therapeutics,* translated by J. B. Yeo, New York, Wm. Wood & Co., 1885, v. 3, p. 547.
4. Norton, N. R. Forced respiration in a case of carbolic acid poison, *Med. & Surg. Rep. Presbyterian Hosp., N. Y.,* 1896, *1*:127.
5. Emerson, H. Artificial respiration in the treatment of edema of the lungs, *Arch. Int. Med.,* 1909, *3*:368.
6. Barringer, T. B. Pulmonary edema treated by artificial respiration, *Arch. Int. Med.,* 1909, *3*:372.
7. Barach, A. L., Martin, J. and Eckman, M. Positive pressure respiration and its application to the treatment of acute pulmonary edema, *Ann. Int. Med.,* 1938, *12*:754.
8. Barach, A. L., Fenn, W. O., Ferris, E. B. and Schmidt, C. F. The physiology of pressure breathing; a brief review of its present status, *J. Aviation Med.,* 1947, *18*:73.
9. Cournand, A. Recent observations on the dynamics of the pulmonary circulation, *Bull. New York Acad. Med.,* 1947, *23*:27.
10. Welch, W. H. Zur Pathologie des Lungenœdems, *Virchow's Arch. f. path. Anat.,* 1878, *72*:375.
11. Luisada, A. A. and Sarnoff, S. J. Paroxysmal pulmonary edema consequent to stimulation of cardiovascular receptors, *Am. Heart J.,* 1946, *31*:270.
12. Barach, A. L. and Swenson, P. C. Effect of breathing gases under positive pressure on lumens of small and medium-sized bronchi, *Arch. Int. Med.,* 1939, *63*:946.
13. Barach, A. L. and Molomut, N. An oxygen mask metered for positive pressure, *Am. Int. Med.,* 1942, *17*:820.
14. Barach, A. L. Physiologically directed therapy in pneumonia, *Ann. Int. Med.,* 1942, *17*:812.
15. Barach, A. L. and Molomut, N. An oxygen mask metered for positive pressure, *Ann. Int. Med.,* 1942, *17*:820.
16. Kernan, J. D. and Barach, A. L. Role of helium in cases of obstructive lesions in the trachea and larynx, *Arch. Otol.,* 1937, *26*:419.
17. Motley, H. L., Cournand, A., Werko, L., Dresdale, D. T., Himmelstein, A. and Richards, D. W. Intermittent positive pressure breathing, *J.A.M.A.,* 1948, *137*:370.
18. Motley, H. L., Werko, L., Cournand, A. and Richards, D. W. Observations on the clinical use of intermittent positive pressure, *J. Aviation Med.,* 1947, *18*:417.
19. Barach, A. L. *Physiologic therapy in respiratory diseases.* 2.ed. Philadelphia, J. B. Lippincott Co. 1948.
20. Dock, W. and Taubman, F. Some technics for recording the ballistocardiograms directly from the body, *Am. J. Med.,* 1949, *7*:751.
21. Molomut, N. and Allen, S. C. Effect of pressure breathing on circulation at high altitude as measured by the ballistocardiograph, *J. Aviation Med.,* 1946, *17*:357.
22. Otis, A. B., Rahn, H., Brontman, M., Mullins, J. and Fenn, W. O. Ballisto-

cardiographic study of changes in cardiac output due to respiration, *J. Clin. Investigation,* 1946, *25*:413.
23. Barach, A. L., Eckman, M. et al. Studies on positive pressure respiration, *J. Aviation Med.,* 1946, *17*:200; 1947, *18*: 139; 252.
24. Motley, H. L., Cournand, A., Eckman, M. and Richards, D. W., Jr., Physiological studies on man with the pneumatic balance resuscitator, "Burns model," *J. Aviation Med.,* 1946, *17*:431.
25. Werko, L. The influence of positive pressure breathing on the circulation in man, *Acta med. Scandinav.,* 1947, supp. *193.*
26. Hardy, G. C. and Barach, A. L. Positive pressure respiration in the treatment of irritant pulmonary edema due to chlorine gas poisoning, *J.A.M.A.,* 1945, *128*:359.
27. Ansbro, F. P. Positive pressure respiration in the treatment of acute pulmonary edema, *Am. J. Surg.,* 1945, *68*:185.
28. Carlisle, J. M. Pulmonary edema, *J.A.M.A.,* 1943, *123*:947.
29. Barach, A. L. The therapeutic use of helium, *J.A.M.A.,* 1936, *107*:1273.

AEROSOL THERAPY IN SINUSITIS, BRONCHIECTASIS AND LUNG ABSCESS

CHESMORE EASTLAKE, JR

Assistant in Medicine, College of Physicians & Surgeons, Columbia University

ANTIBIOTIC aerosol therapy techniques are designed to deposit a nebulin in areas where a close contact can be made with infecting organisms, particularly in regions of the body to which the blood supply does not adequately transport a blood-borne agent to contact bacteria . . . or where there is a selective barrier active to prevent passage of drugs from blood vessels into the area in question. Examples of these areas include the sinuses draining into the nose and the bronchi. Sulfonamide drugs may be found in bronchial secretions in the same concentration as in the blood stream; penicillin, however, does not share this propensity to penetrate into the bronchial lumen. It has been shown that intramuscular injection of penicillin has often failed to alleviate the symptoms of bronchiectasis, whereas subsequent use of antibiotic aerosol has produced good clinical results. The effectiveness of an aerosol depends mainly upon the topical deposition of a quantity of antibiotic sufficient to overcome the pathogenicity of the virulent organisms present.

No attempt will be made in this summary to review the literature on aerosol therapy. The methods and results reported herein have been